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Observational studies

Pain and other symptoms in patients with chronic benign paroxysmal positional vertigo (BPPV)

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HIGHLIGHTS

- Chronic benign paroxysmal positional vertigo (BPPV) is associated with pain.
- Most chronic BPPV patients in the presents study have neck pain (87%), headache (75%), or widespread musculoskeletal pain (40%).
- Most chronic BPPV patients in the present study have fatigue (85%), visual (84%), and cognitive (81%) disturbances.
- The Dizziness Handicap Inventory (DHI)-questionnaire can be a useful tool in identifying treatable balance disorders with pain.

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ABSTRACT

Background and aim: A diagnosis of chronic benign paroxysmal positional vertigo (BPPV) is based on brief attacks of rotatory vertigo and concomitant nystagmus elicited by rapid changes in head position relative to gravity. However, the clinical course of BPPV may vary considerably from a self-limiting to a persisting and/or recurrent disabling problem. The authors' experience is that the most common complaints of patients with chronic BPPV are nautical vertigo or dizziness with other symptoms including neck pain, headache, widespread musculoskeletal pain, fatigue, and visual disturbances. Trauma is believed to be the major cause of BPPV in individuals younger than fifty years. Chronic BPPV is associated with high morbidity. Since these patients often suffer from pain and do not have rotatory vertigo, their symptoms are often attributed to other conditions. The aim of this study was to investigate possible associations between these symptoms and chronic BPPV.

Methods: During 2010 a consecutive prospective cohort observational study was performed. *Diagnostic criteria*: (A) BPPV diagnosis confirmed by the following: (1) a specific history of vertigo/dizziness evoked by acceleration/deceleration, (2) nystagmus in the first position of otolith repositioning maneuvers, and (3) appearing and disappearing nystagmus during the repositioning maneuvers; (B) the disorder has persisted for at least six months. (C) Normal MRI of the cerebrum. *Exclusion criteria*: (A) Any disorder of the central nervous system (CNS), (B) migraine, (C) active Ménière's disease, and (D) severe eye disorders. Symptom questionnaire ('yes or no' answers during a personal interview) and Dizziness Handicap Inventory (DHI) were used.

Results: We included 69 patients (20 males and 49 females) with a median age of 45 years (range 21–68 years). The median duration of the disease was five years and three months. The video-oculography confirmed BPPV in more than one semicircular canal in all patients. In 15% there was a latency of more than 1 min before nystagmus occurred. The Dizziness Handicap Inventory (DHI) median score was 55.5 (score >60 indicates a risk of fall). Seventy-five percent were on 50–100% sick leave. Eighty-one percent had a history of head or neck trauma. Nineteen percent could not recall any history of trauma. In our cohort, nautical vertigo and dizziness (81%) was far more common than rotatory vertigo (20%). The majority of patients (87%) reported pain as a major symptom: neck pain (87%), headache (75%) and widespread pain (40%). Fatigue (85%), visual disturbances (84%), and decreased concentration ability (81%) were the most frequently reported symptoms. In addition, unexpected findings such as involuntary movements of the extremities, face, neck or torso were found during otolith repositioning maneuvers (12%). We describe one case, as an example, how treatment of his BPPV also resolved his chronic, severe pain condition.

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Conclusion: This observational study demonstrates a likely connection between chronic BPPV and the following symptoms: nautical vertigo/dizziness, neck pain, headache, widespread pain, fatigue, visual disturbances, cognitive dysfunctions, nausea, and tinnitus.
Implications: Patients with complex pain conditions associated with nautical vertigo and dizziness should be evaluated with the Dizziness Handicap Inventory (DHI)-questionnaire which can identify treatable balance disorders in patients with chronic musculoskeletal pain.

1. Introduction

Benign paroxysmal positional vertigo (BPPV) was first described by Barány in 1921 [1] and coined by Dix and Hallpike in 1952 [2]. The condition is characterized by "...brief attacks of rotatory vertigo and concomitant positioning rotatory-linear nystagmus elicited by rapid changes in head position relative to gravity" [3]. According to this statement an individual without rotatory vertigo does not suffer from BPPV. However, in 1995 Norré [4] stated that BPPV is not always characterized by rotatory vertigo. The clinical course of BPPV may vary considerably from a self-treatable, self-limiting, to a persisting and/or recurrent disabling problem. Although it is named as such, there are a considerable number of patients who do not perceive it as a benign disease, but rather as an incapacitating condition that restricts their daily activities and has a significant impact on their quality of life [5,6]. Furthermore, it is well recognized that BPPV secondary to trauma is associated with high morbidity [7–12].

Also according to the authors' experience the clinical picture of chronic BPPV can differ from the original descriptions of Dix and Hallpike [2] and Brandt and Daroff [3]. The description cited above is still the most frequently used when describing BPPV [3,13]. However, in patients with chronic BPPV we find the most common complaints are nautical vertigo (sensory illusion reminding of movements experienced on board a ship in waves) and dizziness with various other symptoms including neck pain, headache, and widespread musculoskeletal pain. In addition, they suffer from photophobia, fatigue, visual disturbances (e.g. blurred vision, seeing "stars" of different colors even in darkness), and episodes of tunnel vision lasting a few minutes. Movements of the head evoke all of these associated symptoms. They dislike certain environments that may cause peripheral visual stimulation and provoke symptoms, e.g. busy shopping centers or driving through a tunnel.

Because these patients often complain of pain and do not have rotatory vertigo, their pain symptoms are frequently attributed to other conditions.

The aim of this observational study was to investigate associations between chronic BPPV and the following symptoms: nautical vertigo, dizziness, neck pain, headache, widespread musculoskeletal pain, fatigue, visual disturbances, and tinnitus.

2. Methods

This was a prospective observational study on patients referred to the Otoneurology Centre in Southern Norway during the period April 2010 to January 2011 by general practitioners or other specialists such as ENT surgeons and neurologists. Most patients referred were considered difficult cases due to the complexity of symptoms and their inability to function normally in society. Written informed consent was obtained from all patients. The procedures of the study are in accordance with ethical standards on human experimentation and with the Helsinki Declaration of 1975, as revised in 1983. The Regional Ethical Committee did not consider that a formal approval was necessary.

2.1. Diagnostic criteria

(A) BPPV diagnosis confirmed by the following:
  (1) specific history of vertigo or dizziness evoked by acceleration or deceleration,
  (2) nystagmus in the first position of otolith repositioning maneuvers,
  (3) appearing and disappearing nystagmus during the repositioning maneuvers;
(B) the disorder had persisted for at least six months.
(C) Normal MRI of the cerebrum.

2.2. Exclusion criteria

(1) disorder of the central nervous system (CNS),
(2) migraine (confirmed by positive effect of migraine specific medication),
(3) consequent smooth pursuit eye movement ataxia, i.e. uni or bilateral ataxia characteristic for a CNS disorder (pontine or cerebellar lesion),
(4) saccadic eye movement test disturbance, especially hyper- or hypometria, which are characteristic for a CNS disorder such as multiple sclerosis,
(5) active Ménière's disease,
(6) severe eye disorders, i.e. conditions where optimal vision cannot be obtained by optical aids,
(7) inability to fill in questionnaires.

2.3. Procedure

2.3.1. Dizziness handicap inventory (DHI) [15,16]

This is validated for individuals with vestibular dysfunction. The tool consists of 25 items that are scored as *always* (4 points), *sometimes* (2 points), and *never* (0 point) for a maximal score of 100. A score > 60 indicates an increased likelihood of having a fall.

2.3.2. Symptom questionnaire

"Yes" or "no" answers during a personal interview (shown in Table 1).

The definition of vertigo and of dizziness is according to Stedman's Medical Dictionary (26th edition) [17].

Vertigo is defined as:

(1) "A sensation of spinning or whirling motion. Vertigo implies a definite sensation of rotation of the subject or of objects about the subject in any plane;
(2) imprecisely used as a general term to describe *dizziness* commonly used by patients in an attempt to describe various symptoms such as faintness, giddiness, light-headedness, or unsteadiness".

We chose to operate with either *rotatory vertigo* or *nautical vertigo* (=sensory illusion reminding of movements experienced on board a ship in waves) and *dizziness*.

The visual disturbances the patients report are blurred vision, seeing "stars" of different colors even in darkness, episodes of

Table 1
Frequency of symptoms reported before treatment.

	N = 69	%
Rotatory vertigo	14	20
Nautical vertigo/dizziness	56	81
Headache	50	75
Neck pain	58	87
Widespread pain	27	40
Visual disturbance	58	84
Phonophobia hyperacousis	42	61
Tinnitus	37	54
Peri-retroorbithal pressure/pain	46	67
Sensation of globus	33	48
Nausea	51	74
Temperature disturbances	35	51
Sleep disturbances	46	67
Impaired short term memory	45	65
Concentration problems	56	81
Impaired simultaneous capacity	35	51
Aggravation by physical activity	54	78
Fatigue	59	85

The cipher indicates the number of individuals who has reported the specific symptom. The percent express the frequency of each symptom.

tunnel vision lasting a few minutes, all evoked by movements of the head.

2.3.3. Diagnosing procedure

The diagnosing procedure was performed simultaneously by the two first authors. The nystagmus was registered by video-oculography (Interacoustics A/S, Assens, Denmark) (Fig. 1). Oculography is superior to oculoscopy as it is able to register the lowest amplitude nystagmus.

Otoliths and debris in the semicircular canals (SCCs) are the cause of BPPV, i.e. they are solid particles in fluid.

It is difficult to move solid floating particles in liquid by rapid movements; therefore the repositioning maneuvers are done in slow motion. Because of the possible long latency before nystagmus evokes in some patients, they were kept for 2 min in each position during the diagnostic procedure. Otoliths in one SSC give a specific nystagmus pattern. Otoliths in more than one SSC give various nystagmus patterns according to which SSC is activated and the amount of debris present.

The resulting nystagmus was analyzed like a vector-diagram. Divergence from one-SSC pattern was interpreted as a BPPV with otoliths in more than one SSC [18].

Polensek and Tusa in 2009 found the caloric test unnecessary in diagnosing BPPV [19]. The caloric test and the head impulse test were excluded in this study because they do not give reproducible results in patients with BPPV. This is because the otoliths in the SCC are influenced by the slightest movement and the latency and nystagmus duration are variable. The oculomotor test battery (i.e. smooth pursuit eye movement test and saccadic eye movement test) has been applied to exclude patients with CNS disorders.

2.3.4. Repositioning maneuvers

These were performed with the purpose (in addition to treatment) to confirm the BPPV diagnosis according to point (3) of the diagnostic criteria for BPPV.

2.3.4.1. Epley maneuver for otolith repositioning in posterior SSC [20,21]. Relax in sitting position for 5 min. The maneuver was carried out in slow motion. The subject was kept in each position for 3 min.

2.3.4.2. Otolith repositioning in anterior SSC (Fig. 2). This maneuver is developed by the authors and based on studying a dissected temporal bone. It was carried out in slow motion:

(1) Relax in long sitting position on a tilting table for 5 min. Turn the head 20° toward the unaffected side.
(2) The patient's head is maintained in 20° of rotation and the table is slowly tilted backwards to an angle more than 70° under the horizontal level.
(3) Hold the tilted position and slowly rotate the head 40° to the opposite side.
(4) Finally, the table is tilted back to upright position.

Each position should be maintained for at least 3 min.

2.3.4.3. Modified 360° repositioning maneuver for repositioning of otoliths in the horizontal semicircular canal. The maneuver is also developed by the authors and is based on studying a dissected temporal bone.

(1) Relax in sitting position for 5 min.
(2) Slowly lie down toward the affected side. A pillow should be placed under the patient's head.
(3) Next, chin in and slowly turn into the supine position.
(4) Then turn slowly toward the unaffected side.
(5) Chin in and turn into the prone position (remove the pillow and rest on the forehead).
(6) Finish by going back into the starting (sitting) position.

Each position should be maintained for at least 3 min.

2.3.5. Statistical analysis

The StatSoft-statistical program, copyright 2004 (Tulsa, OK 74104) was used for the analyses. Fischer 2×2 tables was used for comparing the frequency of symptoms during otolith repositioning maneuvers and symptoms reported in the questionnaire.

3. Results

3.1. Patients with the diagnosis of benign paroxysmal positioning vertigo – BPPV

During the nine month, period 203 patients were assessed. Eighty-six individuals fulfilled the diagnostic criteria. All participants were younger than 69 years of age. Five patients were excluded due to an earlier CNS-disorder (three with migraine, two with epilepsy); four were excluded due to active Ménière's disease; two individuals were excluded due to severe eye disorders, and six were excluded due to inability to fill in the questionnaire.

Thus, 69 patients (20 males and 49 females) with a median age of 45 years (range 21–68 years) had BPPV. The median duration of the disease was 5 years and 3 months (range from half a year to 30 years). Of the 69 patients 52 (75%) were on 50–100% sick leave.

3.2. Trauma

Fifty-six patients (81%) had a history of head or neck trauma resulting from road traffic accidents, fall accidents, or sports injuries. Twenty-six patients (38%) fulfilled the Quebec Task Force criteria for the diagnosis Whiplash Associated Disorders (WAD) [14]. Thirty individuals (43%) had a history of trauma before debut of vertigo. The majority of these claimed that their disorder could be related to the trauma. Thirteen (19%) could not recall any trauma. Age, gender, duration of illness, and sick leave were equally distributed among the patients with WAD, with other trauma, and those without recalled history of trauma.

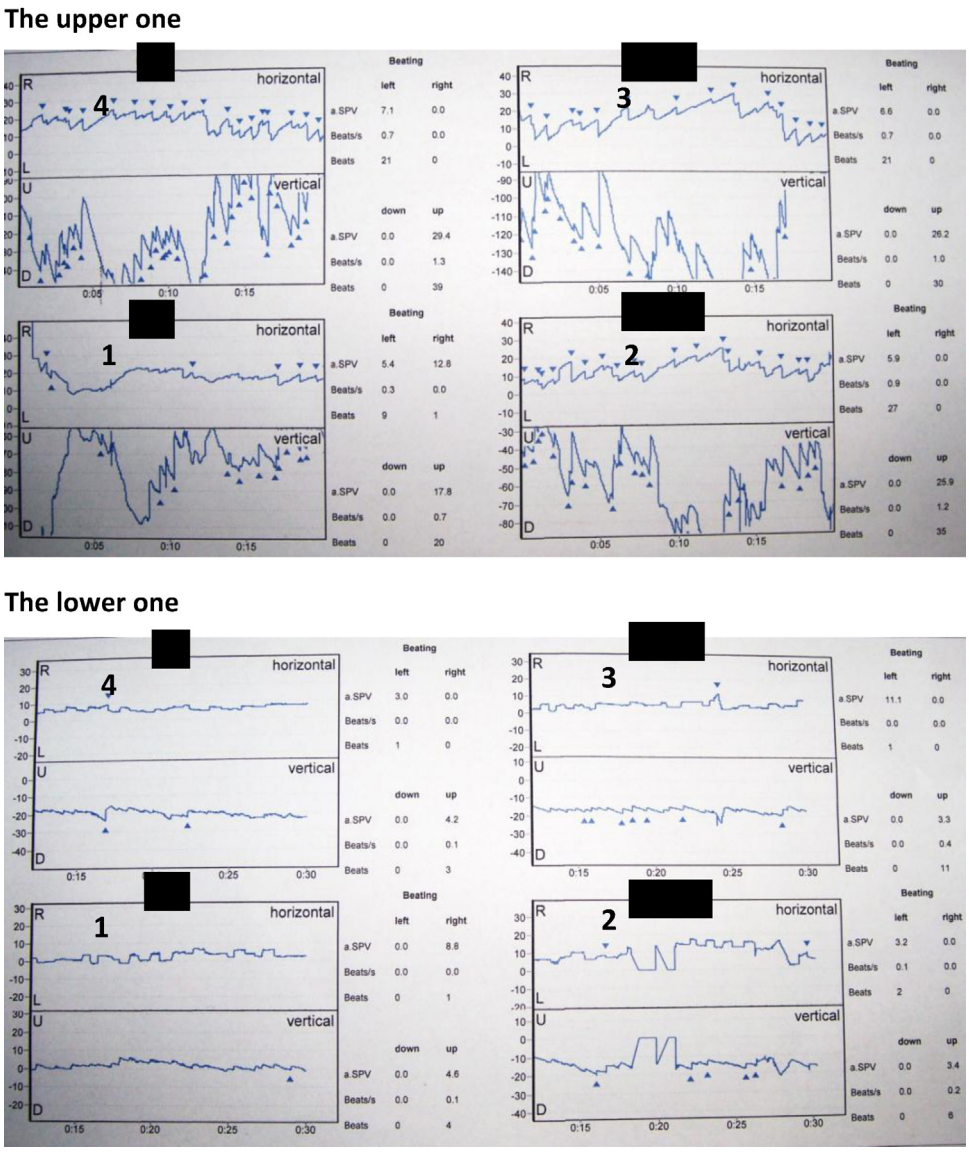

Fig. 1. Video-oculographic documentations of BPPV **pre-treatment** (the upper one) and **post-treatment** (the lower one). Equipment from Interacoustics A/S, Assens, Denmark was used. The patient was tested in the Dix–Hallpike position to the right for 2 min. Each of the four rectangles (1–4) shows the first 20 s of a 30 s period of time. At the right side of each period illustration (1–4) the quantitative details are given, i.e., the counts of nystagmus and their orientation (right, left, up and down) during a 30 s period, beats per second, and SPV, i.e. Slow Phase Velocity of nystagmus beats. The recordings are from a 38 year old man with an eight years history of chronic widespread pain and fatigue after a traffic road accident. His level of pain varied from 5 to 9 on a visual analog scale of pain (0–10). Pain was the absolutely devastating symptom. There was no report of vertigo but dizziness. DHI-score was 60. The test results are shown in *the upper illustration*. The patient underwent treatment of BPPV in the anterior, posterior and horizontal SSCs bilaterally. During a period of one year he improved significantly. During this year he had some relapses, which were treated. In total he received 15 treatments. The only recommendation given as a home-exercise program was walking (hiking) with *gradual increase* in distance and intensity. Exactly one year after the first examination he was reexamined (the lower one) in the Dix–Hallpike position to the right for 2 min. The illustration shows very few nystagmus beats compared with the pre-treatment figure. After this late control the patient has been working full time as a handyman without any complaints.

3.3. Analgesic drugs used

Thirty-four patients (49%) used either NSAIDs together with paracetamol or codeinphosphate, and 18 (26%) used tramadol for pain relief. Sixteen (23%) had stopped using analgesic drugs because they were ineffective. The other 18 patients used their pain medication only occasionally. They all answered that their vertigo/dizziness occurred whether medication was used or not.

3.4. Vertigo

The video-oculography confirmed BPPV in more than one semicircular canal in all patients. In 10 individuals (15%) there was a latency of more than 1 min before nystagmus occurred during the diagnosing procedure.

The Dizziness Handicap Inventory (DHI) median score was 55.5 (range 26–90 points) (Std. dev 16.52).

In these 69 patients, nautical vertigo and dizziness (81%) was far more common than rotatory vertigo (20%), see Table 1.

3.5. Pain

The majority of patients (87%) reported pain as a symptom, i.e., neck pain (87%), headache (75%) and widespread pain (40%).

3.6. Other associated symptoms

Fatigue (85%), visual disturbances (84%), as well as decreased ability to concentrate (81%) were the most frequent reported symptoms. Thirty-five patients (51%) (8 males and 27 females) suffered

Table 2
Symptoms reported during otolith repositioning maneuvers for the posterior-, anterior- and horizontal semicircular canals.

	N = 69	%
Rotatory vertigo	17	25
Nautical vertigo/dizziness	52	75
Pain	47	68
Nausea	35	51
Visual disturbance	31	45
Change of tinnitus intensity	32	46

The cipher indicates the number of individuals who has reported the specific symptom. The percent express the frequency of each symptom.

from temperature disturbances. Eight females were below forty years of age.

3.7. Repositioning maneuvers

Patients' symptoms reported during the otolith repositioning maneuvers are listed in Table 2. In addition to the nystagmus (100%), involuntary movements of the extremities, face, neck, or torso (tremor-like, athetotic-like or hemiballism-like) were observed during otolith repositioning maneuvers (12%). Simultaneously with these involuntary movements intense hyperactivity in the eye muscles was registered. During repositioning most symptoms, apart from nausea ($p < 0.01$) and visual disturbances ($p < 0.01$), appeared with the same occurrence as reported in the questionnaire, i.e., rotatory vertigo, nautical vertigo and dizziness, pain, and change in tinnitus intensity (n.s.) (Tables 1 and 2).

4. Discussion

4.1. Nautical vertigo and dizziness, pain and other associated symptoms

Nautical vertigo and dizziness are more common than rotatory vertigo in patients with chronic BPPV. We found that nautical vertigo and dizziness, neck pain, headache, widespread pain fatigue, visual disturbances, and cognitive difficulties are the main symptoms of chronic BPPV.

Their symptoms have previously been attributed to other conditions such as basilar migraine, cervical dizziness, whiplash associated disorders (WAD), fibromyalgia, phobic postural vertigo, and chronic fatigue syndrome.

Furthermore, this study highlights that the majority of patients were on long term sick leave. This is a significant burden on both the individual and on society as a whole [6].

A diagnosis of BPPV is considered confirmed when all three criteria were fulfilled: a specific history of movement related vertigo and dizziness; nystagmus in a BPPV evoking position; as well as nystagmus that appears and disappears during the otolith repositioning [4].

Most reported symptoms in the questionnaire occurred with the same frequency as during the repositioning maneuver. This supports our claim of an association between the non-vestibular symptoms described and a chronic BPPV.

4.2. Migrainous vertigo, cervical dizziness, phobic postural vertigo, anxiety, and chronic fatigue syndrome

However, von Brevern et al. [22] claimed that 10 subjects from a cohort of 362 patients with positional vertigo suffered from migrainous vertigo mimicking BPPV. They have migrainous symptoms (e.g. visual disturbances) and atypical positional nystagmus. In our study all patients have atypical nystagmus due to involvement of more than one SCC [18].

Moreover, sixty percent of the patients in our study experienced visual disturbances. Therefore, we raise the question could the 10 patients of von Brevern et al. [22] with suspected migrainous vertigo instead be suffering from a chronic BPPV?

The existence of the diagnosis of "cervical dizziness" is debatable. Clinical studies on cervical dizziness have two weak points: (1) the inability to confirm the diagnosis and (2) the unexplained discrepancy between patients suffering from severe neck pain without vertigo and patients complaining of disabling vertigo with only moderate neck pain [23].

If phobic postural vertigo, anxiety, and chronic fatigue syndrome can provoke dizziness, they can definitely not elicit the nystagmus related to the BPPV canalith repositioning maneuvers [24].

Dizziness is a frequent side-effect in most pain medication. The majority of patients reported to have used medication sporadically. Vertigo and dizziness occurred whether medication was used or not.

4.3. Importance of long latency of provoked nystagmus

Fifteen percent of our patients with BPPV had a latency of more than 1 min before nystagmus occurred. Therefore, the diagnosis could be missed, if the patients are observed for half a minute in the test positions.

Patients with long latency have been sick for a long period. In analogy with the teleological purpose of immobilization in the anti-inflammatory response we consider a parallel attempt to immobilize free floating otoliths in the endolymph to obtain stability. This could possibly be achieved through increased viscosity by secretion of colloids.

4.4. Importance of nautical vertigo and pain for the diagnosis of BPPV

The two highest ranked symptoms observed are nautical vertigo and dizziness and pain. Therefore, we suggest that the ". . .brief attacks of rotatory vertigo and concomitant positioning rotatory-linear nystagmus elicited by rapid changes in head position relative to gravity" [3] cannot be used to diagnose chronic BPPV. It is noted that most patients suffer from pain, such as neck pain, headache, and widespread pain. It is assumed that this pain is caused by the chronic BPPV since treatment according to these principles has been successful in reducing the patients' pain [25].

4.5. The balance dysfunction in a BPPV is a dynamic and not a static one

Afferent information from the vestibular, visual and somatosensory systems converges in multiple areas within the CNS and is important for general equilibrium, body orientation, and oculomotor control. Abnormal afferent input from these systems can result in abnormal postural control.

Resulting mismatch which may occur in the presence of conflicting afferent information is thought to underlay symptoms of dizziness or unsteadiness [26].

In a stable condition of impaired vestibular function, e.g. after a labyrinthectomy, the vestibular nuclei (VN) complex compensates this loss by adjusting facilitating and inhibitory activity of the contralateral VN complex [27], i.e. a static balance dysfunction.

It is impossible to compensate in the same way within a BPPV with its dislocated free-floating otoliths and debris, because the affected labyrinth(s) transmit(s) varying abnormal signals from time to time to the same stimuli. This is possibly why their symptoms are ongoing.

Thus in BPPV, the VN complex receives contradictive signals from the different receptors of the labyrinths. The VN complex is

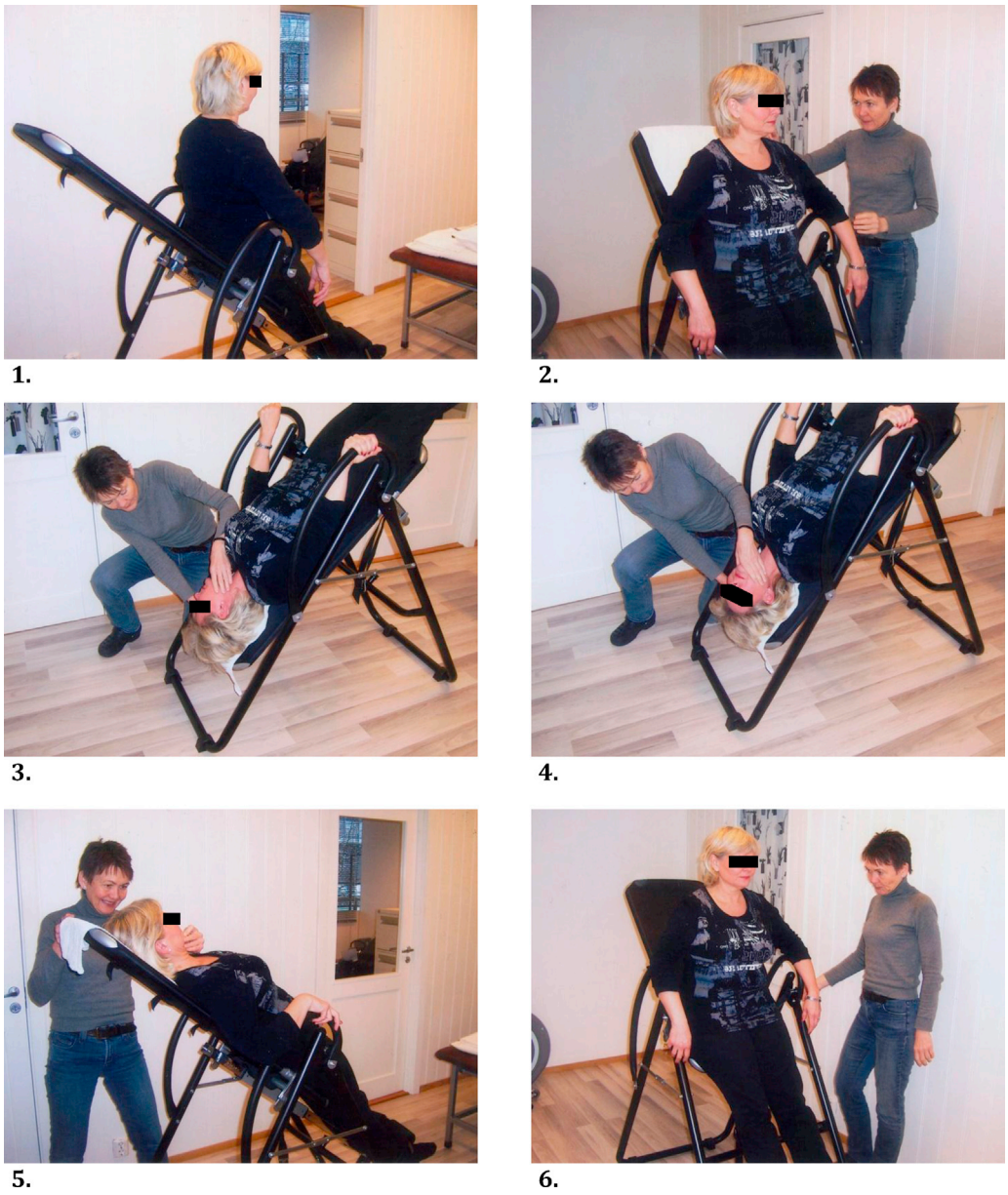

Fig. 2. Repositioning of otoliths in anterior semicircular canal (right side). (1) Relax in long sitting position on a tilting table for 5 min. (2) Rotate the head 20° toward the unaffected side. (3) Slowly move the patient backwards until the head is at least 70° under the horizontal line. Hold this position for 3–5 min. (4) Hold the tilted position and slowly rotate the head 40° to the opposite side (i.e. the affected side). Hold this position for 3–5 min. (5) Finally, the table is tilted back to upright position in slow motion. (6) Stay in the upright position for 5 min before, if necessary, repeating the maneuver. The patient sits still for at least 15 min after treatment is completed.

the origin of the different vestibular reflexes: the vestibulo-ocular reflex, the lateral and medial vestibulo-spinal reflexes as well as the vestibuloreticular reflex.

4.6. Headache

The varying contradictive signals through the vestibulo-ocular reflex in patients with BPPV are the cause of visual disturbances [28]. Hypertonus in the intra- and periorbital muscles is a part of the explanation of the frontal headache [29].

4.7. Neck pain and generalized musculoskeletal pain

The postural control is secured through vestibulo-spinal [30–33] and vestibulo-reticular reflexes [34].

The teleological purpose is to secure vertical balance. The vestibulo-spinal reflexes are involved in control of neck, torso and extremity muscle tension. The reticular formation maintains a level of tonus and integrates information from several neural centers.

Postural mismatch in BPPV occurs in all situations when the otoliths in the semicircular canals are activated, i.e. when the head is moved. There are individual strategies to compensate for this postural mismatch. The most common way to compensate a dynamic balance disturbance seems to be static use of muscles. It is well documented that static use of muscles generates accumulation of pain generating substances, i.e. arachnoid acid, bradykinin, and histamin [35]. Pain leads to lack of motor control, i.e. a sensorimotor control disturbance [36,37]. Furthermore, according to O'Sullivan et al. [38] proprioceptive deficit may lead to delayed neuromuscular protective reflexes and coordination such that muscle contraction occurs too late to protect the joint from excessive joint movement, and possible damage of articular surfaces through repetitive abnormal loading.

Furthermore, these deficits lead to a destructive abnormal movement pattern.

There is a very high density of gamma-muscle spindles in the cranio-cervical joint related deep muscles [39–41]. These are the most important muscles in delivering proprioceptive signals. The proprioceptive information from muscle spindles is crucial for optimal motor control [42,43].

Therefore, according to O'Sullivan et al. [38] it seems reasonable to presume that improper proprioceptive activity would lead to impaired postural control and pain. This is consistent with the observed high frequency of neck pain in our study. There is evidence to suggest that the vicious circle of the equilibrium control system is the motor for the vicious circle of pain.

4.8. Fatigue and involuntary muscle movements

The high frequency of fatigue is a consequence of the above-described continuously working postural control system. A disturbance in the delicate integration of facilitatory and inhibitory signals in the myostatic spinal reflexes [44] might explain the observation of rhythmic involuntary movements of neck, face, torso or extremities during otolith repositioning.

Simultaneously, involuntary rhythmic intense eye muscle movements were registered. A few patients had the ability to control the involuntary neck, face, torso, or extremities movements for a short period by focusing straight ahead. The pathway of the smooth pursuit eye movement goes via the inferior olive [45]. This may possibly indicate a connection between the semicircular canals and the olivocerebellar system. The inferior olive acts as a generator of temporal patterns, and acts as a pacemaker with a rhythmical firing [46,47].

A disturbed vestibulo-reticular activity has the potential of creating various symptoms since the paramedian pontine reticular formation (PPRF) has a moderating influence on several functions [34].

4.9. Cognitive deficits

Over the last decade, there has been a steady accumulation of evidence to suggest that vestibular lesions may also lead to cognitive deficits, including deficits in attention, learning and memory [48].

From PPRF there are pathways to the hippocampus, which are responsible for short-term memory, concentration ability, and simultaneous capacity [49].

4.10. Temperature disturbances and intensity of tinnitus

The reported temperature disturbances are not gender or age related. Most likely they are due to activation of the autonomous nervous system. The acoustic symptoms were found to be more common than previously recognized [50,51]. The proprioceptive effect on tinnitus can be explained through afferent proprioceptive activity to the dorsal cochlear nucleus and rostrally in the central auditory system [52,53].

4.11. Treatment of BPPV may resolve most associated symptoms, such as pain

We illustrate how the specific treatment of BPPV will resolve the associated pain condition in Fig. 2. An ongoing prospective treatment study on patients with chronic BPPV will follow in a separate publication.

5. Conclusions

This study demonstrates a likely connection between chronic BPPV and the following symptoms: Nautical vertigo and dizziness, neck pain, headache, widespread pain, fatigue, visual disturbances, cognitive dysfunctions, nausea, and tinnitus.

The balance dysfunction in BPPV is dynamic and not static. This leads to a perpetual postural mismatch. Thus, the vicious circle of the equilibrium control system is the motor for the vicious circle of pain. Specific treatment of the BPPV may resolve many of the associated symptoms, which is focus for a separate publication.

Conflict of interest

The authors declare that they have no conflict of interest.

Authors' contributions

The first two authors have equally been involved in examination and treatment of all patients. Both authors have equally contributed to conception, design, and acquisition of data, analysis and interpretation of data. Moreover, both of the two first authors have equally been involved in drafting the manuscript and have given final approval of the version to be published. The third author has contributed to design and discussion. He has given the final approval of the version to be published.

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